cholinergic reflex arc. This hypothesis is especially attractive because it is known that inhaled sulfur dioxide activates tracheobronchial irritant receptors that in turn stimulate the afferent arc of the cholinergic reflex. All sulfites are converted to sulfur dioxide in solution. The mechanism of susceptibility to metabisulfites in these patients, however, remains obscure.

Metabisulfite sensitivity probably is not rare. A history of anaphylaxis occurring following ingestion of restaurant food and drink, particularly salads and avocado dip, suggests that metabisulfite sensitivity reactions have occurred. Anaphylaxis after ingestion of soft drinks with sulfur dioxide or wines; shrimp or other seafood, or restaurant potatoes or vegetable combinations sprayed with potassium metabisulfite solutions should alert a physician to the possibility of this syndrome.

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Urticarial Vasculitis

URTICARIA IS CHARACTERIZED clinically as pruritic, erythematous, cutaneous elevations arising from dilated blood vessels and areas of cutaneous edema. After intradermal injection, many mediators of inflammation are capable of producing lesions that resemble urticaria. These mediators include histamine, the anaphylatoxins of complement (C3a, C4a and C5a), certain leukotrienes, some prostaglandins and perhaps serotonin.

Inflammation within blood vessels is called vasculitis and may appear as the consequence of one or more mechanisms. As detected by routine histologic and immunofluorescence techniques, vasculitis is frequently found in acute urticaria precipitated by adverse drug reactions, hepatitis B infection or serum sickness due to heterologous protein administration. Vasculitis is less frequently observed in the specimens from skin biopsies of patients with chronic idiopathic urticaria. However, recent investigations clearly show characteristic histologic or plasma complement abnormalities, or both, in some patients with chronic idiopathic urticaria.

Although mild inflammatory changes of vasculitis are detected in the specimens from skin biopsies of many patients with chronic idiopathic urticaria, the patients with chronic urticaria and necrotizing venulitis and others with hypocomplementemic vasculitic urticarial syndrome constitute distinct subsets of urticarial patients with more prominent systemic features and more severe vasculitis. Necrotizing venulitis was observed in adults with recurring urticaria, episodic arthralgias or arthritis, abdominal pain or glomerulitis. The plasma complement analyses in some of these patients indicated activation of the classical pathway of complement as shown by decreased total hemolytic complement (CH_{50}), Clq, C4 and occasionally C3. In other patients the complement analyses were normal.

The other syndrome, called the hypocomplementemic vasculitic urticarial syndrome or systemic lupus erythematosus (SLE)-related syndrome, was observed in adult women who have persistent urticaria, leukocytoclastic angiitis, severe angioedema, arthralgias, arthritis, neurologic complications, sensitivity to potassium iodide and profound hypocomplementemia. The complement analyses of these patients' plasmas showed depressed CH_{50}, C4, C2 and C3 in association with deficiency of Clq. The deficient Clq apparently relates to the presence of an unusual immunoglobulin that precipitates Clq (7S Clq precipitin or low molecular weight Clq precipitin). Both the Clq deficiency and the 7S Clq precipitin are characteristic of this syndrome.

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Rhinitis

INFLAMMATION OF THE nasal mucosa is commonly termed rhinitis. There are a number of causes. A diagnostic classification can be based on more specialized nasal evaluations including nasal cytology, rhinomanometry, nasal challenge, and olfactory and ciliary function testing.

Nasal cytology is carried out by gently scraping the mucosal lining of the inferior turbinate with a calcium-alginate applicator and placing the specimen on a glass slide. The prepared slide is then metachromatically stained and the cell pattern examined microscopically. Nasal airway resistance

TABLE 1.—*Classification of Nasal Disorders Based on Systematic Evaluation*

Inflammatory rhinitis
Eosinophilic allergic rhinitis
seasonal
perennial
Eosinophilic nonallergic rhinitis
Infectious rhinitis
viral
bacterial
Nasal polyps
Nasal mastocytosis
Atrophic rhinitis
Noninflammatory rhinitis
Vasomotor rhinitis
associated with local or systemic condition
autonomic dysfunction
Rhinitis medicamentosa
Structure-related rhinitis

can be measured by an active, single-nostril anterior rhinomanometric technique. Simultaneous recording of pressure and flow rate allows calculation of resistance. This determines the degree of reversible and fixed nasal obstruction when done before and after topical administration of a decongestant. This technique can also be used to record changes in nasal patency following allergic or chemical challenges and drug therapy. To evaluate for anosmia or hyposmia, the threshold for detecting inhaled pyridine is determined. Ciliary function is most simply tested after placing a small particle of saccharin on the anterior portion of the inferior turbinate. Elapsed time is recorded until a sweet taste is noted by the patient. Structural and functional abnormalities of cilia will prolong the time interval from placement to taste.

On the basis of this systematic evaluation, nasal disorders can be classified, as shown in Table 1. It should be noted that many of these categories can coexist, and may be acute or chronic in nature.

Organizing an approach on which to base clinical and laboratory findings can assist us in understanding these disorders and direct our continued research into their pathogenesis. Such an approach can place each rhinitis patient in a functional classification that suggests specific therapy. Similar methods have assisted the development of diagnostic and treatment criteria for lower airway diseases. Thus we can anticipate progress in the care of patients with rhinitis.

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Seminal Fluid Allergy

URTICARIA AND HYPERVENTILATION after coitus have occasionally been reported to physicians, who attributed such reactions to psychic agitation. However, a case has been reported of a woman who had anaphylactic shock consisting of angioedema, wheezing, "uterine pain" and cardiovascular collapse within minutes of coitus. A glycoprotein fraction of seminal plasma was identified as the antigen. Diluted to 10^{-6} it caused a positive reaction on direct skin tests; her serum caused a positive reaction on passive transfer tests. Since this initial case there have been sporadic reports of anaphylaxis due to semen. Levine and co-workers reported the case of a patient who had anaphylaxis, positive findings on skin tests and leukocyte histamine release (LHR) with a 20,000 to 30,000 molecular weight (mol wt) fraction of seminal plasma, but not of sperm extract. Her leukocytes reacted with this fraction of semen from seven different men tested.

IgE-antibody involvement in such cases has been found by radioallergosorbent test (RAST) with seminal fluid fractions. We have seen a patient who three times had anaphylaxis necessitating epinephrine; she also had a concomitant severe flare of her eczema. Immunotherapy with a dilution of her husband's semen has been attempted. Condom usage by the patient's husband has prevented subsequent reactions, except on two episodes of "condom tear accidents" when she had localized urticaria. RAST and LHR values fell during absence of exposure, only to rise abruptly after each accident.

Of four patients reported by Bernstein and associates, two had anaphylaxis that required epinephrine and two had only genital tissue edema and burning pain. Both women with anaphylaxis had positive reactions on skin tests, RAST and LHR. One had her first episode on first coitus after childbirth, as did our patient; a disrupted vaginal mucosa may add risk to such sensitization. The two women with severe localized reactions had positive findings on lymphoproliferation and leukocyte migration inhibition assays with both sem-